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Subinvolution of the Placental Site

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UNDER normal circumstances an efficient primary hemostatic mechanism operates following expulsion of the placenta and limits blood loss ordinarily to less than 500 ml. While there may be insignificant further loss in the lochia rubra, any notable bleeding after the immediate postpartum period is regarded as abnormal. Probably the most common origin for such bleeding is the placental site, and this paper is concerned with the histologic findings at the placental site in 100 cases of delayed postpartum bleeding.

Studies of normal hemodynamics in the gravid uterus have been published by Romney and associates,¹ and detailed investigations of the vascular patterns in myometrium, endometrium and placenta by Ramsey,² and will not be reviewed here.

The precise day-by-day sequence of histologic events in postpartum involution of placental site vessels is not clearly defined. The normally involuting placental site is not usually disturbed, and specimens for study infrequently encountered. The combined studies of Wormser,³ Goodall,⁴ Teacher,⁵ Williams,⁶ and Sharman⁷ include less than 50 uteri examined at all stages of the puerperium. Following completion of the third stage of labor, the severed ends of the decidual blood vessels undergo thrombosis. Over a period of time these thrombi organize, the placental site becomes smaller, the endometrium regenerates, and the

integrity of this area becomes reconstituted; the endometrium away from the placental site is regenerated by three weeks, but the placental site itself does not fully involute until six or seven weeks postpartum.⁶ It is difficult to draw a base line in the absence of more definite information, and, particularly during the first week or ten days postpartum, it is often clinically impossible to distinguish between normal and delayed involution. Moreover, in the past, attention has been directed more to endometrial regeneration^{6, 8} than to alterations in blood vessels.

Likewise, the literature relating to subinvolution of the placental site (or non-involution, as it is often called) is not abundant. Evans⁹ was the first to associate vascular damage with subinvolution of the placental site. Rutherford and Hertig,¹⁰ reporting three cases, called attention to the failure of complete physiologic obliteration of placental site vessels in the absence of retained trophoblastic tissue and discounted the role of the chronic inflammation in the genesis of subinvolution. Of the six cases reported by Gainey, Nicolay and Lapi,¹¹ four patients developed postpartum hemorrhage at six and seven days postpartum, and the histologic features, on review, seem inconclusive. However, their two patients who bled on the 11th and 18th postpartum days showed typical lesions. Retained trophoblastic remnants were not found, and hemorrhage was attributed to vascular lesions. Greene¹² reported a single case of subinvolution with bleeding on the 10th postpartum day. Paalman and McElin¹³ summarized relevant clinical data in 38 patients with delayed postpartum hemorrhage and no retained trophoblast. Sixty per cent of their cases required curettage in the second week following delivery; 11 of their patients were primiparae and 27 multiparae. A recent report by Salm and Simons¹⁴ deals with two patients with recurrent, intractable bleeding developing several months after delivery, each requiring hysterectomy after two attempts to control hemorrhage by curettage had failed. From these and other reports we can develop the clinical picture associated with subinvolution of the placental site as delayed postpartum hemorrhage, abrupt in onset, occurring at any time from one week to several months after delivery, maximally during the second week of the puerperium; on examination the uterus is larger than expected, soft and boggy; the cervical os is patulous and blood trickles or pours from it.

A more elaborate study of the problem of late postpartum hemorrhage by Wolfe and Pedowitz¹⁵ divides the cause into three major

TABLE I—AGE DISTRIBUTION IN 100 CASES OF SUBINVOLUTION OF THE PLACENTAL SITE

Up to 20 years	8
21-25 years	21
26-30 years	27
30-35 years	25
36-40 years	10
Over 40 years	5
Age not stated	4
	<hr/> 100

groups: 1) retained placental fragments; 2) abnormality in involution of the placental site; 3) abnormality in retention and separation of decidua vera. They point out that a retained placental fragment may be spontaneously expelled, give rise to one or more episodes of bleeding necessitating surgical removal, or become enmeshed, forming a placental polyp. They distinguish between retained placental elements and abnormal involution, but do not assign a cause to the latter. Heath¹⁶ reviewed 100 cases of delayed postpartum hemorrhage occurring over an eight-year period in one hospital. Seventy-five per cent of the patients were multiparae. Study of the curetings obtained when the patients were re-admitted for hemorrhage showed placental tissue in 33 per cent of the cases; a diagnosis of subinvolution, however, was made in only eight per cent of the cases.

MATERIAL, METHODS, AND CLINICAL DATA

Part of the material in this study was collected at the Armed Forces Institute of Pathology and some cases have been added from the files of the Boston Lying-in Hospital, supplemented by cases seen in consultation. In no sense is this series consecutive, nor is it to be construed as representing a sample of a specific obstetrical population. In addition to clinical records, pathologic material consisting of curetings and hysterectomy specimens were examined. Tissue sections stained routinely with hematoxylin and eosin supplemented by such special stains as Masson's trichrome technique, elastica-van Gieson, and Schiff's periodic acid method form the basis for most of the observations made.

TABLE II—INTERVAL BETWEEN ABORTION OR DELIVERY AND ONSET OF DELAYED POSTPARTUM HEMORRHAGE IN 100 CASES OF SUBINVOLUTION OF THE PLACENTAL SITE

Less than 12 days	8
12 days-21 days	16
22 days-30 days	15
1 month-2 months	24
2 months-4 months	21
Over 4 months	5
Interval not stated	11
	—
	100

A total of 100 cases was studied. The patients ranged from 17 to 46 years in age (Table I); 40 per cent of the patients were over 30 years of age. Of 66 patients in whom gravidity was recorded, 15 were primigravidae (23 per cent), and 51 were multigravidae (77 per cent). It is evident that the lesion is more common in women who are older than the average obstetrical patient and in those who have borne more than one child. Fifty women had been delivered at or near term; 45 of these could be considered to have had a normal term delivery, including one set of twins; three had been delivered by cesarean section; one patient had been delivered by hysterectomy at 26 weeks for placenta previa; one patient had been delivered of twins at 35 weeks and her immediate postpartum course had been complicated by uterine inertia. Apart from these last two cases, no evidence in the antepartum history could be found which might indicate previous uterine disease. Manual removal of the placenta had been necessary in several cases, but the use of this maneuver was not systematically recorded in the available clinical data. Forty-two women developed delayed hemorrhage following an abortion; in each of these there was an interval of one week or more in which bleeding was absent. Two of these postabortal cases had aborted hydatidiform moles. There was a conspicuous incidence of unsuspected abortion in this group, as well as a sprinkling of cases in which abortion had been procured. In eight patients the type of antecedent gestation could not be determined from the records available.

The interval between the date of delivery or abortion and the date

of onset of delayed postpartum hemorrhage merits comment (Table II). Accurate data were available in 89 of the 100 cases. In eight cases the interval was less than 12 days, in 16 cases 12 to 21 days, in 15 cases 22 to 30 days. In 24 cases uterine bleeding developed one to two months following delivery or abortion; in 26 cases the interval was from two to over four months. Thus, in over 50 per cent of the cases, hemorrhage occurred after the puerperium was presumably completed.

In 59 cases curettage was the primary method of treatment, and ergonovine was administered postoperatively in varying doses for varying periods to almost all of these patients. This was successful in 54 patients. Two patients developed such brisk bleeding after curettage that emergency hysterectomy was done, and in one case the uterus was perforated and removed. In another two cases a second curettage was necessitated when bleeding recurred after a symptom-free interval; in neither of these two patients was the second curettage successful; again serious bleeding recurred after an interval, and hysterectomy was done (Salm and Simons).¹⁴ In 41 cases hysterectomy without prior curettage was the primary method of treatment. This group of patients includes 13 in whom postpartum hemorrhage developed after a term delivery, often with a long latent period, 17 after abortion, including several cases in which the abortion was not suspected, eight cases in which the fate of the previous gestation was not stated, and three cases in which the hemorrhage developed two to four months following repeat cesarean section. Subtracting these last three cases, and six cases in which hysterectomy was indicated because of other intrinsic uterine or ovarian disease, as well as five cases in which hysterectomy was done for reasons known only to the surgeon, we find 27 cases in which hysterectomy was done as a primary procedure of choice.

PATHOLOGIC OBSERVATIONS

Gross examination of curetted material revealed little information of value; and gross examination of surgically removed uteri showed, in a minority of specimens, only a small, irregular patch of altered endometrium variously described as roughened, discolored, and occasionally hemorrhagic. The important observations were found on microscopic examination.

Examination of curettings, as well as the endo-myometrium of hysterectomy specimens, showed the involuted placental site to con-

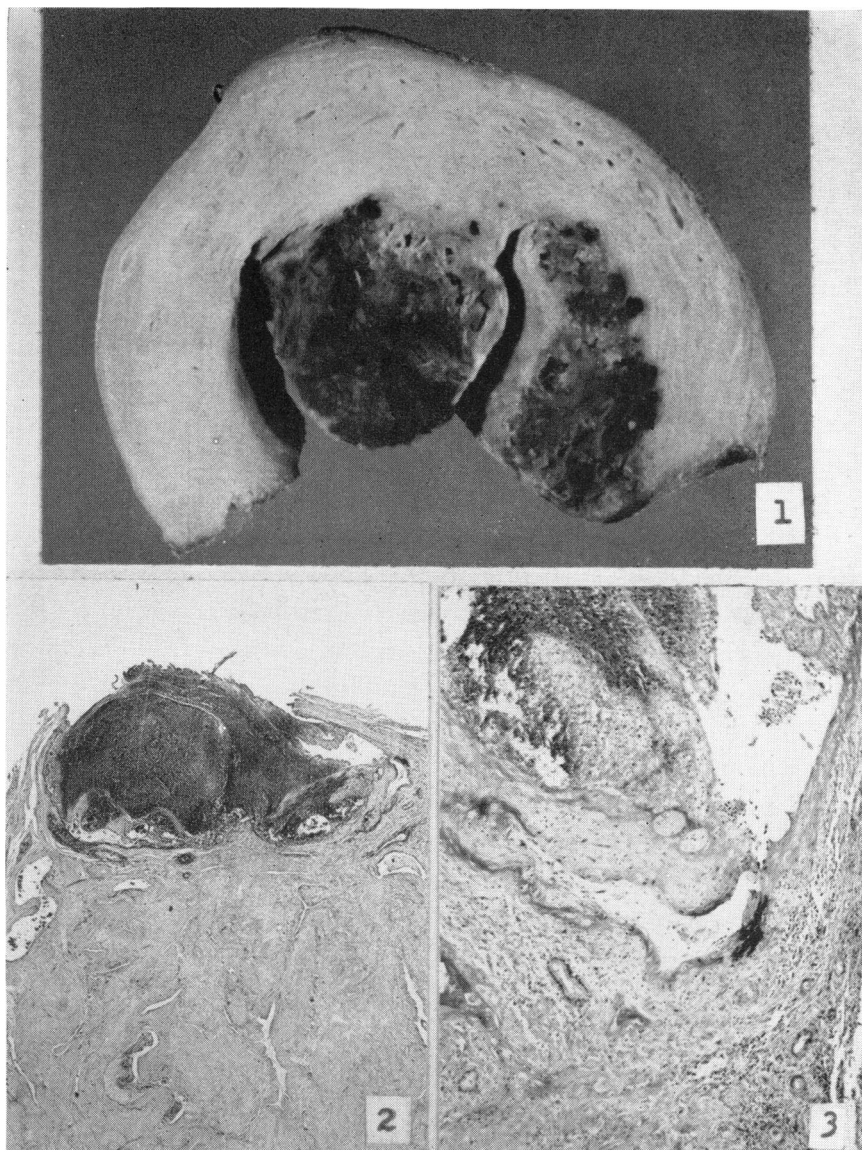


Fig. 1: Supracervical hysterectomy 6 weeks postpartum showing a "placental polyp" formed by a retained accreted cotyledon. The discolored area to the right shows subinvolved vessels, some of which are thrombosed, extending half way through the thickness of the uterine wall.

Fig. 2: Hysterectomy 10 weeks postpartum demonstrating a superficial lesion, readily accessible to the curet, composed of a large thrombus in a distended vascular space. (From the Armed Forces Institute of Pathology, Washington, D.C., Picture No. 53-7761 acc 576681)

Fig. 3: Detail of Fig. 2, the lower right-hand part of the lesion. Two ghost villi are seen. Regenerated endometrial glands are present. Fibrinoid material is present in the vessel wall. (From the Armed Forces Institute of Pathology, Washington, D.C., Picture No. 53-7760 acc 576682)

tain an admixture of amorphous debris, thrombotic material, degenerating decidua, and regenerating endometrium, in varying proportion. Leukocytic infiltration of varying degree and composition was almost constant; and in many cases hemosiderin phagocytosis was conspicuous. It is difficult to gauge the "appropriateness" of the degree of endometrial regeneration in respect to the number of days following evacuation of the products of conception; endometria of both proliferative and secretory type were found and bore no relationship to any temporal parameter. However, the regenerated endometrium was not wholly normal in most cases; glandular tortuosity was often exaggerated and cystic dilatation of glands was not infrequently seen. The bulk of the inflammatory exudate was located in the regenerating endometrial stroma, along with patches of decidua undergoing degeneration both by cytolysis and by hyalinization and hemosiderin-laden macrophages.

Specimens obtained during the first 12 postgestational days showed relatively scanty endometrium. Parenthetically, six of the eight specimens in which the diagnosis of subinvolution of the placental site was made during the first 12 postgestational days were hysterectomy specimens; it is extremely difficult to make this diagnosis on the basis of curettings alone during the early period of involution. A number of cases of delayed postpartum hemorrhage during this period were examined and discarded from the study because of the difficulty in deciding whether the lesions were sufficiently characteristic; the lack of a precise knowledge of the day-by-day range of normal alterations found in the placental site makes the determination of a base line hazardous. From this period only those cases were accepted in which the deviation was so egregiously *praeter naturam* that the diagnosis was forced upon one. (Figures 1 to 3.)

The significant lesion common to all of these cases was an alteration in blood vessels, presumably arterial in nature, but so modified that in many instances it was difficult to decide whether they had originally been small arteries or veins. The vessels were large, dilated and tortuous, and thick-walled. During the early postgestational stage they tended to form an agglomeration suggestive in pattern of a cavernous hemangioma, but as time progressed the individual vessels became separated and more distinct as the regenerating endometrium separated their coils. In curettings, it was often difficult to ascertain the level of the blood vessels; but fragments of underlying myometrium removed by the

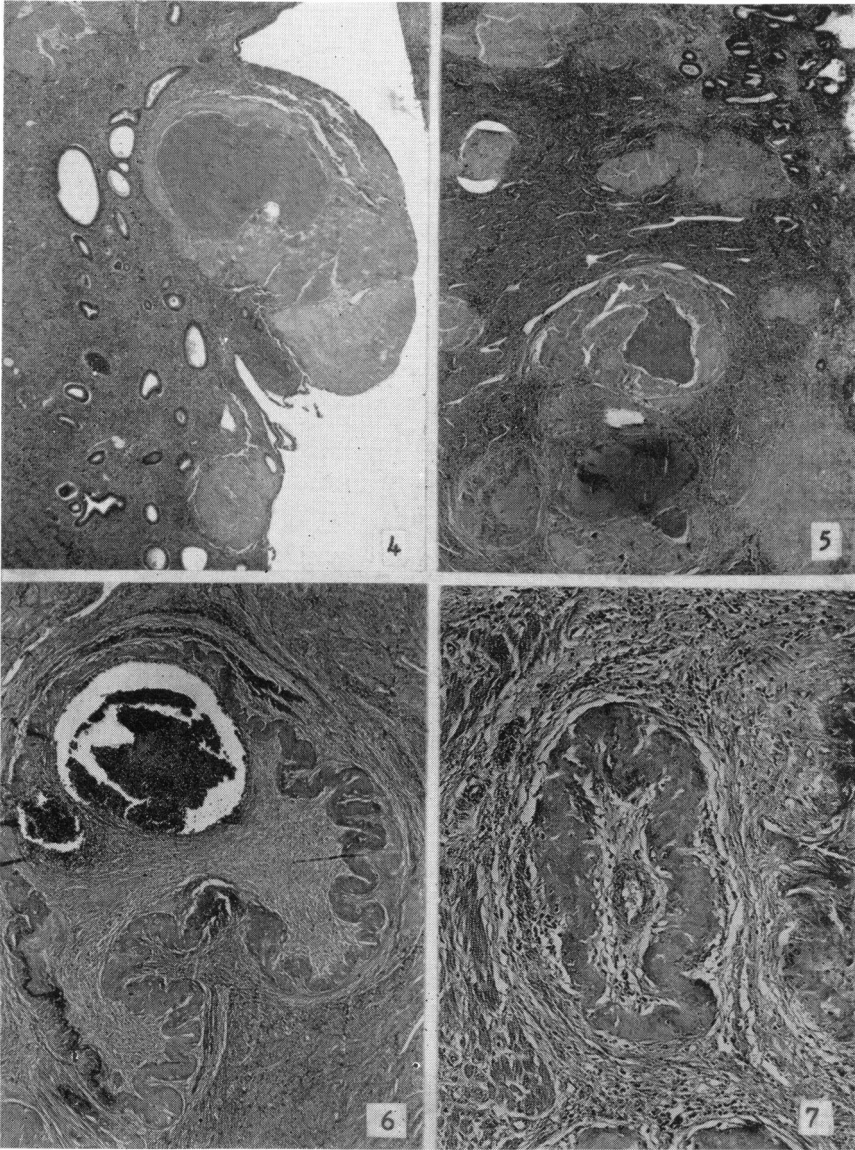


Fig. 4: Hysterectomy 10 weeks postpartum. The endometrium is well regenerated. A single large vessel representing an enormously distended and thickened spiral arteriole is the source of bleeding. To its right is a small patch of hyaline material representing an imperfectly involuted arteriole which is not a source of bleeding.

Fig. 5: Hysterectomy specimen 4 weeks postpartum illustrates regenerated endometrium and a number of vessels at the level of radial and arcuate arteries. Some are merely occluded and hyalinized; others are patulous, containing thrombi.

Fig. 6: An arcuate artery showing the thick, hyalinized, scalloped media. Only a small segment of internal elastic lamella has been preserved. Proliferated spindle cells fill most of the lumen, and there is an open channel with an eccentric thrombus which is not organized and only loosely adherent. Curettage at about 6 weeks; hysterectomy at 9 weeks for recurrent bleeding. Focal placenta accreta found. Elastica-van Gieson stain.

Fig. 7: A radial artery with hyalinized scalloped media, intimal proliferation, and a newly formed central vascular canal. Masson trichrome stain. Same case as Fig. 6.

curet often showed lesions which were located in arcuate arteries. These large vessels were usually open, sometimes filled with conglutinated erythrocytes, less frequently with lamellated thrombotic material, and occasionally with some degree of organization of such thrombotic material. In fortunately oriented specimens some of these damaged vessels were seen to communicate with the endometrial surface, and it is not difficult to conjecture that bleeding may have ensued following dislodgement of a small, unorganized thrombus from its distal portion. In a few cases, only a single vessel showed subinvolution.

The thickening of the vessel walls was due to deposition of hyaline material which replaced much of the muscular coat of the vessel. The hyaline material was not uniformly eosinophilic and glassy; occasionally it was fibrillary in texture, even "fibrinoid". It is plausible to infer that hyalinization represents homogenization of an alteration in the ground substance of vessel walls. In material from early cases the amount of hyaline was not large. It becomes conspicuous after the 12th postpartum day and increases in prominence over the next two weeks, by which time it is fully developed. Admixed with the fibrillary and hyaline material was often a collar of large, pale cells surrounded by an intercellular material which was PAS-positive. We believe these cells are decidual in nature, and support this interpretation by the observation of peripherally located PAS-positive material within their cytoplasm and by their pavement-like arrangement when seen in sections transecting the vascular wall obliquely. The hyalinized vessels often assumed a scalloped contour. Completely involuted vessels were often represented by a scalloped hyaline body revascularized by a small central vessel, the configuration suggesting nothing quite so much as a corpus albicans in the innermost layers of myometrium. However, in subinvolved vessels the central cavity of the scalloped hyaline spherule is patent; it may contain an eccentric zone of fibroblastic proliferation, patches of organizing thrombus, or even a central channel filled with uncoagulated blood. Not uncommonly, all three forms may be present in one field. Likewise, in older specimens it was not uncommon to find completely involuted placental site vessels adjacent to imperfectly involuted ones; the former with their tiny central vessels could not possibly have served as a source for hemorrhage; the latter, by virtue of their size, partial patency, and location, almost surely are. (Figures 4 to 7.)

Sections stained by Masson's trichrome method revealed clearly the

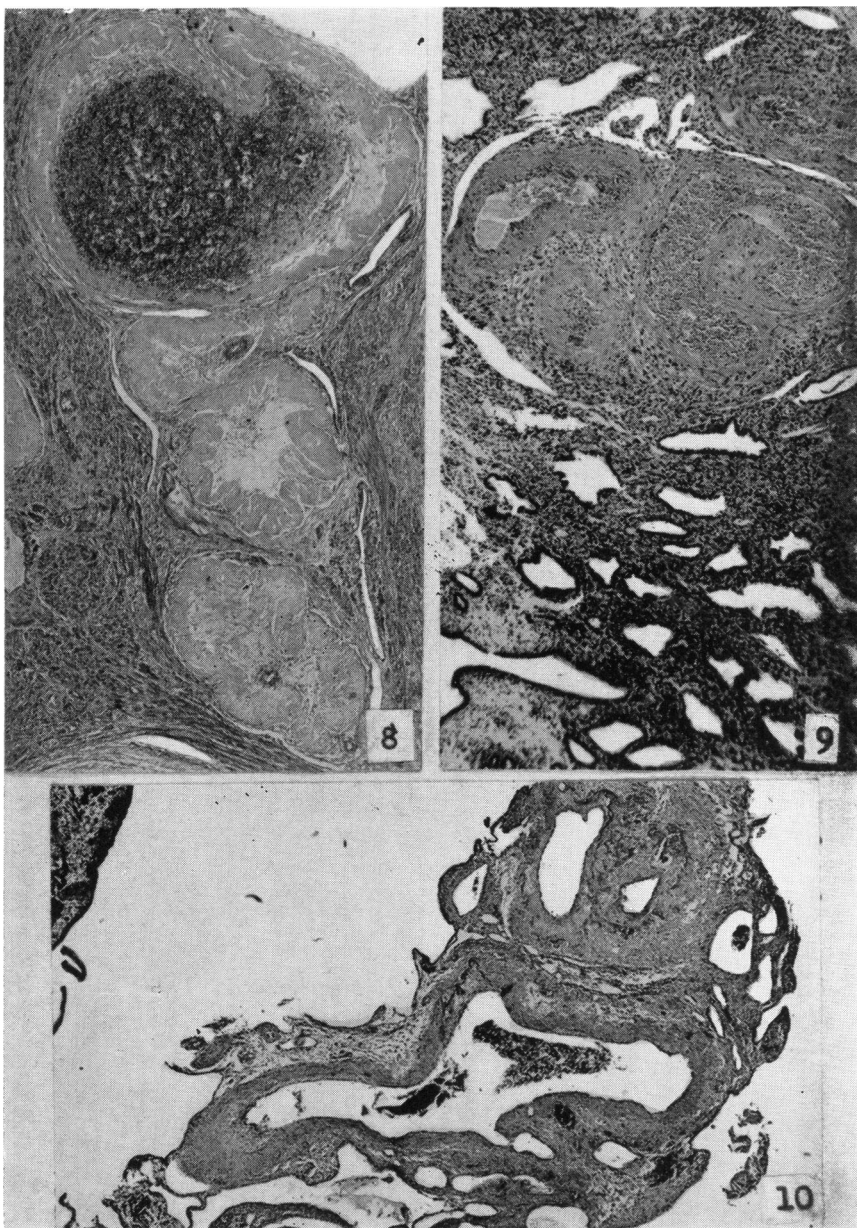


Fig. 8: A tortuous vessel at the arcuate-radial level cut in several planes. Some portions are occluded and show the "vessel-within-a-vessel" pattern. One segment is patulous and contains partly organized thrombus. Same case as Fig. 6.

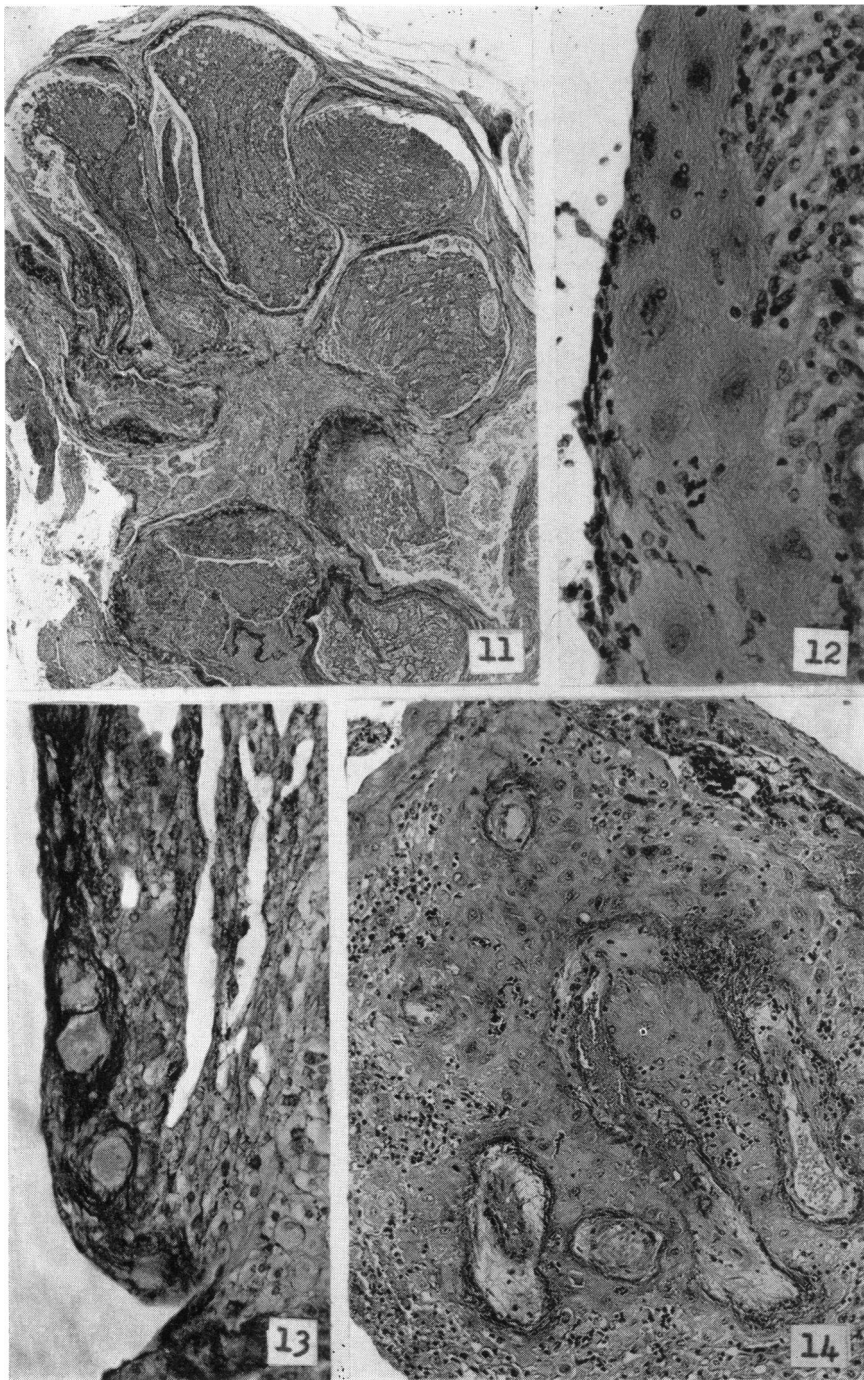
Fig. 9: Uterine curettings 6 weeks postabortal showing a distorted spiral arteriole in a regenerated endometrium with irregular glandular contours. The arteriolar wall is hyalinized and contains polyhedral pale cells. This was the only abnormal vessel found in extensive sectioning of the curetted material. No retained trophoblast. (From the Armed Forces Institute of Pathology, Washington, D.C., Picture No. 53-6720 acc 575309)

Fig. 10: A basal and spiral arteriole in uterine curettings 12 weeks postabortal showing distortion and ectasia. The endometrium is regenerated and contains a few cystically dilated glands. No retained trophoblast.

fibroblastic nature of the intimal proliferation in vessels held partly patent. Section stained with the elastica-van Gieson method showed that in early specimens, up to about three weeks postgestation, the elastica was absent, or failed to react in the usual way to selective stains. It reappeared slowly and patchily after this time and its staining properties reverted to normal. The formation of the "vessel-within-a-vessel" pattern, i.e., a newly formed vessel with its own intima and media inside a pre-existing vessel with its own intima and media preserved, takes about six to eight weeks to develop to such a point that it is unmistakably identifiable.

The material was carefully examined for evidence of retained placental tissue. In material in which no placental elements were found on primary examination, deeper sections of paraffin blocks were made and additional tissue embedded. Multiple sections were made through the placental site in most of the hysterectomy specimens, and in most of the cases in which curettings were the only material available, all the curettings were embedded. In the 100 cases, retained placental tissue was found in 53 instances, but in 47 cases there was no microscopic evidence of retained placental tissue. When these data were analyzed in relation to whether the antecedent gestation had terminated by abortion or by term delivery, it was found that in the 42 cases of delayed postabortal bleeding, retained placental elements were present in 27 cases (64 per cent) and absent in 15 cases (36 per cent). This relationship was reversed in cases of delayed postpartum bleeding; in these 50 cases, placental relics were found in 19 cases (38 per cent) and absent in 31 cases (62 per cent). In the eight cases in which information was lacking as to whether the antecedent gestation had terminated by abortion or by term delivery, placental tissue was found in seven cases. (Figures 8 to 10.)

The form assumed by retained placental elements varied considerably. In one case the major portion of a cotyledon had been retained and was firmly adherent to the uterine fundus, forming a placental polyp. Large blood vessels near the margin of the polyp showed typical changes of subinvolution. In other instances, a small cluster of viable chorionic villi was attached to the superficial layers of myometrium, being embedded in a fibro-hyaline matrix in which decidua were absent, deficient, or in the form of a few scattered cells; it is difficult to distinguish sharply between this configuration and that seen in a microscopic



(Legend appears on next page)

focus of placenta accreta. In a few of such examples, retained chorionic villi were actually accreted to the walls of thickened blood vessels, and in two such cases even penetrated into the vascular lumen. In still other cases, only an occasional chorionic villus was seen, often in the form of a ghost villus surrounded by thrombotic material. In many instances it was evident that the trophoblast surrounding retained chorionic villi was well preserved and viable; however, in an approximately equal number its viability was doubtful, and in a few cases only villous stroma surrounded by a thin rim of eosinophilic material was identified.

Retained placental tissue in the form of gross polyps down to small aggregates does not undergo organization and vascularization from maternal tissue. It remains attached to the uterus, but it does not participate in the reaction of the uterus. Park¹⁷ has suggested that the failure of granulation tissue to invade retained placental tissue may be related to the properties of a homograft reaction. (Figures 11 to 14.)

In the two cases in which the lesion developed following passage or evacuation of a hydatidiform mole, placental site giant cells were numerous, and buds of syncytiotrophoblast were seen scattered throughout the regenerating endometrium and subjacent myometrium, approaching the configuration seen in syncytial endometritis. Of the 27 cases in which retained placental tissue was found following an abortion, the occurrence of antecedent abortion had not been clinically suspected in six cases; these patients had not received the benefit of immediate postabortal curettage. Also, in three other cases, abortion had been procured and pregnancy interrupted without careful and complete curettage. These nine cases out of 27 (33 per cent) suggest that meticulous curettage following abortion will materially reduce the

LEGEND

Fig. 11: Uterine curetting 4 weeks postpartum showing a fragment of tissue with numerous cavernous spaces filled with thrombi. The remainder of the curetted tissue showed no vascular abnormalities. No retained trophoblast.

Fig. 12: Detail of Fig. 10 showing decidual-like cells forming a collar just below the endothelium.

Fig. 13: Parallel section to Fig. 12. A strongly fuchsinophilic intercellular material is seen between the decidual-like cells in the arteriolar wall. Decidual cells in the surrounding stroma contain granules of similar tinctorial properties. Periodic acid-Schiff stain.

Fig. 14: Persistence of decidual reaction around a spiral arteriole 4 weeks postabortal. Retained trophoblast was absent in this case. Masson trichrome stain.

occurrence of delayed postabortal hemorrhage, but may at the same time increase the potential danger of local instrumental trauma.

In general, those cases in which placental tissue was found, and particularly those in which it was found readily and in sizable quantity, developed uterine bleeding within the first month. However, exceptions were noted; the longest interval in a case of retained secundines was 63 days. There was no particular distribution, in point of time, for the appearance of uterine bleeding in cases in which the vascular lesion was found in the absence of retained placental relics; however, the greatest frequency was during the period from two to six weeks after gestation had been terminated.

DISCUSSION

In an era when so much attention is focussed on hemorrhagic diseases in which there is no vascular lesion, it is refreshing for pathologists to study a visible vascular lesion which is associated with hemorrhage. The observation that this vascular lesion tends to occur in an older than average group of obstetrical patients is consistent with the general observation that vascular disease increases with age, but no further inference can be drawn from this point. Likewise, the somewhat increased occurrence of this lesion with multigravidity suggests that vascular damage or imperfect involution in a previous pregnancy may facilitate its development and overt clinical manifestation in a subsequent pregnancy. This might be particularly significant if it could be demonstrated that the implantation site in the most recent pregnancy was the same as in a previous pregnancy, but there is no evidence to confirm this possibility. Indeed, the lesion occurs in primigravidae and in young women with some frequency, and undue emphasis should not be placed on these tendentious observations.

There is no reliable evidence to indicate the pathogenesis of the lesion we have described. It is possible to conjecture that alterations in the ground substance of vessel walls¹⁸ and persistence of decidua in vessel walls furnish the starting materials upon which the thickening and hyalinization develop. Also, it is not difficult to imagine that a vessel with a thickened wall in which changes in elastic tissue and progressing hyalinization are taking place side by side might not be so retractable as a normal vessel nor so readily sealed off by thrombosis. However, our lack of detailed information regarding the minute change

in normal involution of the placental site precludes more than conjecture along these lines. The role of fibrinolysins, either local or systemic, in dislodging imperfectly formed or non-organized thrombi from such vessels, thereby precipitating the clinical episode of bleeding, should be considered; but again, evidence is lacking to establish the role of this mechanism. It is tempting to infer that retained syncytiotrophoblast may furnish the necessary progestational steroids which would promote the persistence of a perivascular decidual transformation and that degenerating trophoblast may furnish local fibrinolysins, but the vascular lesion occurs almost as frequently in the absence of demonstrably retained placental tissue as in its presence. It would be reasonable to assume that retained trophoblast is not the only mechanism for maintaining decidua and that fibrinolysins, if active, could be derived from another source.

There are a few practical observations to be made about the diagnosis of subinvolution of the placental site in the pathology laboratory. First of all, the diagnosis is extremely difficult, perhaps impossible, to make with certainty during the first ten days or even two weeks postpartum. This is almost invariably true if the material for diagnosis consists of curettings alone. The difficulty is chiefly due to our lack of precise information regarding the range of variability of normal involution. In addition, during this stage the development of hyaline in vessel walls may not be great, and the vessels may appear to be more dilated and engorged than structurally altered. To some extent diagnostic difficulty may be less in hysterectomy specimens during the early postpartum period, but it is wise to be conservative unless the lesion is striking. In later specimens the lesion may be confined to one or two small blood vessels. These can only be found if all curetted fragments are embedded; sometimes it is helpful to make sections at different levels in paraffin blocks. Often a useful clue is the appearance of hemosiderin-laden macrophages in a regenerating endometrial stroma; when this is observed, meticulous search for altered blood vessels has sometimes been rewarding.

It should be emphasized that the mere presence of hyalinized blood vessels is not of itself sufficient for the diagnosis of subinvolution of the placental site. So far as our material permits evaluation, hyalinization is one of the normal structural features of involuting placental site vessels. It is the combination of a patulous vascular lumen with imper-

fectly or partially organizing thrombosis within a vessel, the wall of which is distorted by hyaline material which constitutes the lesion.

Another practical point concerns terminology. Material from this class of patients is usually sent to the laboratory with a brief note reading either "retained secundines" or "postpartum bleeding" or "recent abortion, now bleeding". Only rarely does the curettor supply accurate chronological data. There seems to be a psychologic block among obstetricians and gynecologists to inform pathologists about such important features as the date of the last menstrual period, the date of abortion or delivery, the date of a previous curettage, etc. Not infrequently, the pathologist examining such material will emphasize in his report the presence or absence of retained placental tissue or trophoblastic elements. Cases are signed out under such coding labels as "endometritis, non-specific, postpartum or postabortal" or "endometritis, non-specific, consistent with recent delivery or abortion" or "regenerating endometrium with inflammatory reaction" or "retained placental tissue, postpartum" or "retained chorionic villi, postabortal"—in short, useful descriptive terms which indicate the patient's recent clinical status and the physiologic inflammatory and reparative reaction seen at any placental site whether involution is normal or not. It is our firm belief that if the characteristic vascular lesion can be demonstrated, it is more important to stress the fact that the delayed postpartum hemorrhage can be ascribed to abnormally involuting blood vessels, and it is our practice to use the term "subinvolution, placental site", postabortal or postpartum as indicated.

Certain clinical observations merit re-emphasis. First, the lesion can occur following either an abortion or a term delivery. In fact, Evans' case⁹ occurred following a spontaneous second trimester abortion. Second, the delayed hemorrhage may be quite a late phenomenon. In our series, 50 per cent of the cases developed sudden, unexpected bleeding one month or longer after delivery or abortion, and over 25 per cent two months or longer. In a few cases the interval was over four months. In the two striking cases reported by Salm and Simons¹⁴ the initial hemorrhage occurred six months after delivery in one case and nine months after abortion in the other.

In a communication by pathologists, references to therapeutic considerations must needs be diplomatic. It is of interest that in 54 of 59 patients treated by curettage and supportive measures, no further diffi-

culties were encountered. It is, therefore, somewhat disturbing to note that in 27 patients hysterectomy was the primary method of surgical therapy, a figure which seems abnormally high even with due recognition that occasionally curettage is insufficient to control hemorrhage.

CONCLUSIONS

1. Subinvolution of the placental site is commonly associated with delayed postpartum or postabortal hemorrhage. It tends to occur in the older group of obstetrical patients and in multiparae.

2. The important lesion is vascular and consists of thickening and hyalinization of arterial walls at the site of implantation in the regenerating endometrium. The lesion may occur whether retained placental tissue is present or absent. It is speculated that the hyalinization occurs secondary to alteration in the ground substance of the vessel wall and persistence of the decidual transformation around it. Retained placental elements are more commonly found in cases of delayed hemorrhage following abortion than in cases following term delivery.

3. Over half the cases developed postpartum hemorrhage one month or more after termination of gestation; it can be a late complication.

4. Diagnosis may be difficult during the first two weeks. This difficulty, as well as uncertainty as to pathogenesis, can be ascribed to lack of evidence concerning the day-by-day sequence of events and the range of variation in normal involution of the placental site.

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DISCUSSION

PHILIP GOLDSTEIN: Where hysterectomy was done and where placental tissue was not found in the uterus, was any attempt made to find out if there were persistent corpora lutea of pregnancy or lutein cysts which may have contributed to the subinvolution?

WILLIAM B. OBER: An ovary was removed from each of two patients, each specimen containing a sizable papillary serous cystadenoma. The surgeons apparently had the good grace not to remove ovaries indiscriminately from these young women, and I cannot say much about the ovaries as I did not see them. Apparently, they did not see anything to suggest a persistent corpus luteum gestationis. Usually this structure involutes by six or eight weeks postpartum. In the absence of any grossly visible lesion which would impel a surgeon to remove an ovary, I think we can minimize that as a possible factor.

PHILIP GOLDSTEIN: But you are not sure?

WILLIAM B. OBER: Not having seen the ovaries, I cannot say that I am.